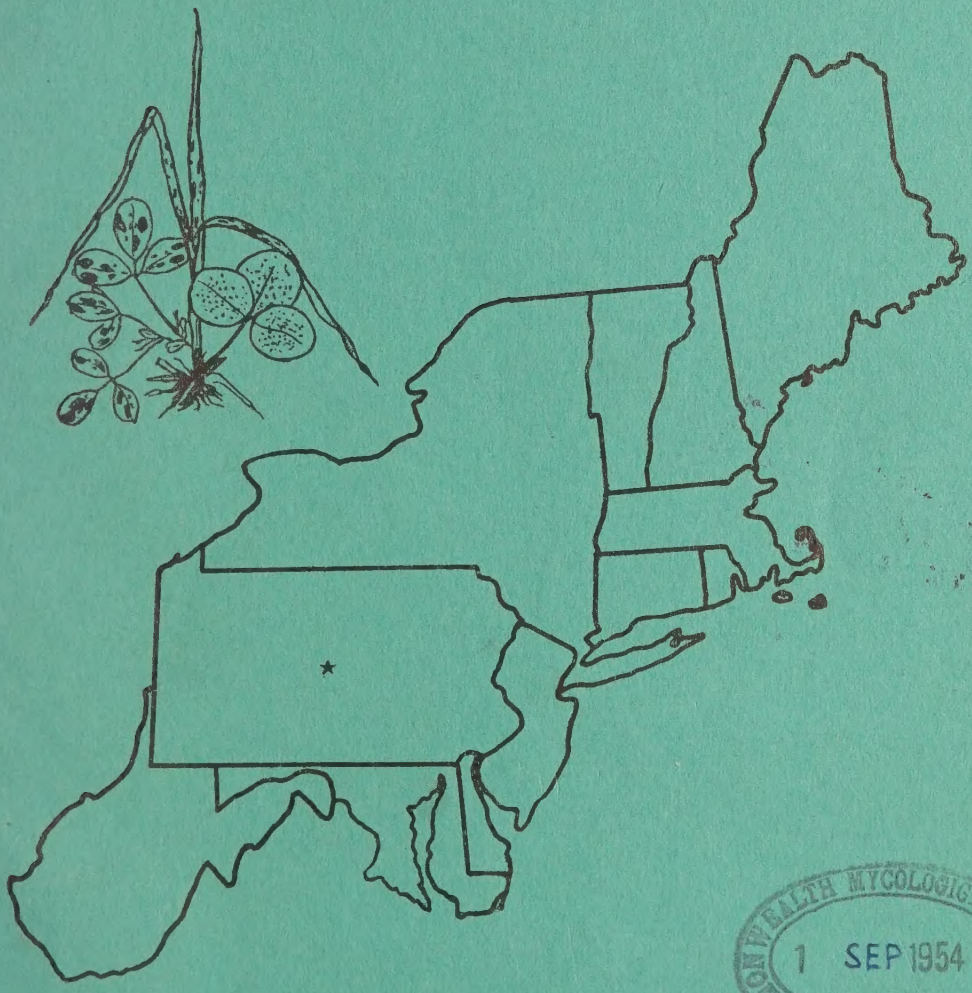
Diseases of Forage Grasses and Legumes In the Northeastern States



The Pennsylvania State University
COLLEGE OF AGRICULTURE
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FOREWORD

With the intensification in the use of grasses and legumes on farms of the northeastern states, the problem of controlling diseases has become more and more an important factor. This, the fifth in the special series of bulletins concerned with grasslands, is designed especially to help identify some of the more important diseases and, in some cases, to suggest helpful means of control. Practically all of the diseases reported herein have been found in Pennsylvania.

The U. S. Regional Pasture Laboratory is located at the Pennsylvania Agricultural Experiment Station, and Pasture Laboratory staff members work in close cooperation with Station research workers on forage problems.—M. A. Farrell, *Director of the Pennsylvania Agricultural Experiment Station.*

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Diseases of Forage Grasses and Legumes In the Northeastern States*

K. W. Kreitlow, J. H. Graham and R. J. Garber†

PRODUCTION OF MILK AND MEAT in the United States is a multi-billion dollar enterprise. Forage, in the form of pasture or hay, is the foundation that supports this enterprise. According to agricultural statistics,[1] there were 88,062,000 head of cattle valued at $15,733,051,000 on farms in the United States in 1952. Of this number, 6,525,000 head, having a value of $1,431,335,000, were being fed from some 30 million acres of grassland in the Northeast.

Dairy cattle in the northeastern states produce annually more than 20 billion pounds of milk, valued at more than one billion dollars. In 1951, cash receipts to farmers of the Northeast from livestock and livestock products totaled some $2,500,000,000. It is not surprising that an acre of good pasture has a minimum value of 200 to 300 dollars. When we further consider that livestock derive more than half of their nutrients from pasture and hay it is evident that growing forage itself is a billion-dollar business.

Any factor, such as plant disease, that reduces yield or quality of forage thus can cause an annual loss running into millions of dollars. This is readily visualized when one considers that a single foliar disease, Pseudopeziza leafspot of alfalfa, may cause 30 per cent or more of the leaves to fall prematurely; yet this is only one of more than 20 diseases that attack alfalfa in the Northeast. Some ailments such as root rots or virus diseases which reduce yields by only a small percentage in any one season may, however, frequently weaken plants so that other organisms can attach and kill then.

So-called "running out" of stands of perennial grasses and legumes often can be traced to attacks by diseases which build up progressively from one season to the next. This is illustrated by the effect of the bacterial wilt disease on susceptible varieties of alfalfa.

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[1]U. S. Department of Agriculture, Agricultural Statistics 1952.

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Very little evidence of the disease is noticeable in seedling stands, but by the third or fourth year stands are frequently so thinned that they are no longer profitable. In addition to reducing yields, diseases such as leaf rust or Stagonospora leafspot of orchardgrass cause premature dying of leaves which appreciably affects quality of forage.

With few exceptions, the diseases discussed in this bulletin occur generally throughout the northeastern states. Many are distributed over most of the United States and some are nearly world-wide. Their prevalence depends on many factors, including presence of susceptible host plants, favorable temperature and moisture conditions, and, for some virus diseases, the presence of suitable insect vectors.

All parts of the plant, root or seed, and plants in all stages of growth from seedling to mature plant are susceptible to attack by one or more of the fungi, bacteria, or viruses. Some disease-producing organisms live for long periods in the soil, others on the residue of plants killed by disease. Still others remain within diseased, living plants which then become reservoirs of infection for neighboring plants.

Although resistant varieties are at present the best means of controlling disease, no such varieties exist for many of the most destructive diseases. Good farm practices—crop rotation, clean cultivation, plowing under old, diseases plant material—help reduce incidence of disease.

Since vigorous plants are better able to withstand attacks of pathogens, adequate soil fertility should be maintained. If foliar diseases strike early and severely, it is best to harvest the stand earlier than normally to remove as much of the diseased material as possible. These general control measures, in addition to those mentioned for specific diseases, help protect forage plants from disease attacks. Much remains to be done in developing more effective means of control.

ALFALFA

Bacterial Wilt (*Corynebacterium insidiosum*)

Bacterial wilt was for many years the most destructive disease of alfalfa in the United States. In parts of the Northeast, particularly the dehydrating area in eastern Pennsylvania, the pathogen is widespread in the soil, while in other parts of the region infested areas are scattered. The disease is favored by abundant moisture and is usually most severe in low, poorly drained spots in alfalfa fields.

Bacterial wilt causes stunting and yellowing of the entire plant. Shortened stems result in bunchy growth, leaves being small, chlorotic and often cupped, fig. *1*. During warm, dry weather plants may wilt and die rapidly. At first only the tips of stems droop, then more or less complete wilting and dying ensues. Stunting is most evident

Fig. 1.—Bacterial wilt of 6-year-old alfalfa.

during regrowth following cutting and progressively less growth is produced after each cutting. Severely diseased plants usually do not survive the winter.

Alfalfa wilt can be positively identified by examining the tap root. When the bark immediately below the crown is peeled a yellowish or pale brown discoloration of the outer wood, in contrast to the creamy-white appearance of healthy wood, characterizes infection. In recently infected plants the yellow stain extends downward from the crown in streaks which merge into a continuous ring beneath the bark. If the tap root is cut across with a sharp knife, scattered discolored dots or a dark ring of discolored tissue is usually visible (*20*), fig. 2.

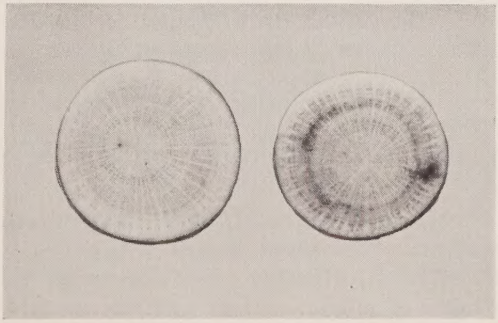


Fig. 2. — Cross-section of healthy and bacterial wilt-infected roots of alfalfa.

The bacteria enter the plant through wounds produced by winter injury or by mechanical injury through harvesting or grazing. The water-conducting tissues are invaded and clogged by rapidly multiplying bacteria, which results in yellowing, stunting, and eventual death of the plant. In advanced stages of the disease, bacteria multiply in the crown and stems and are released into the surrounding soil water.

Use of resistant varieties of alfalfa offers the only practical means of controlling wilt. Resistant varieties, Ranger and Buffalo, are available and a new variety, Vernal, with even greater resistance, is being increased for release to growers. In addition, the grower should follow cultural practices which may aid in maintaining diseased stands and retarding disease development in new areas: (a) maintain high soil fertility levels, particularly potassium and phosphorous; (b) prevent injuries to crown and roots; (c) mow when plants are dry; (d) avoid contaminating young healthy stands, by mowing them before the older diseased fields; (e) do not reseed old alfalfa fields until complete rotting of the infected roots has occurred; (f) do not seed fields that receive surface drainage from infested areas.

Black Stem (*Ascochyta imperfecta*)

Black stem occurs commonly in the United States, Canada, and Europe, and is often severe during periods of cool, wet weather. The fungus attacks leaves, petioles, and stems, frequently causing defoliation and death of plant parts. The organism causing this disease reduces yield and quality of forage and may diminish yields of seed.

In early spring, small brown to black spots develop on the stems and leaves (Plate I, No. 1). Young lesions on stems are at first outlined by a watery border. During the growing season they enlarge and coalesce until much of the stem is blackened. If the plants are left to mature the fungus also may attack pods and seeds. From late fall until spring numerous raised, brown to black fruiting bodies (pycnidia) are visible on the dead stems and petioles. The irregular lesions on the leaves increase in size and may become lighter brown and somewhat zonate. Diseased leaves turn yellow and soon wither and fall (*31*).

The fungus overwinters principally on plant residue. In early spring spores exude from the pycnidia and are splashed by rain onto new shoots. The fungus is carried within the seed and in soil, thus providing other sources of infection.

If severe primary infection occurs in the spring, early harvesting to remove the diseased growth helps to reduce secondary infection. In addition, attempts are being made to develop disease-resistant strains of alfalfa.

Pseudopeziza Leafspot (*Pseudopeziza medicaginis*)

Pseudopeziza or common leafspot, one of the most destructive foliar diseases of alfalfa, occurs throughout the world. Although plants are not killed by the disease, defoliation causes loss of vigor and reduces hay quality and yield. In general, the second and third hay crops are damaged more than the first. During cool, wet weather only the topmost leaves may escape infection, particularly if the plants are growing slowly (*19*).

Small, circular brown spots approximately $\frac{1}{16}$-inch in diameter develop on the leaflets. Brown streaks radiating from the margins of the spots can be seen with a magnifying glass. When spots are fully developed, the centers become thickened and a tiny, light brown, cup-shaped fruiting body (apothecium) forms. This distinguishes the lesions from those caused by black stem. Apothecia are produced on the upper surface of green leaves throughout the growing season, except during prolonged dry weather. Generally, only one apothecium develops in each lesion (Plate I, No. 2).

Spores are discharged from the apothecia and are carried by wind to other plants. The fungus overwinters on dead, undecayed leaves. It is believed that the pathogen is not seedborne.

Though adapted varieties of alfalfa are only moderately resistant to *Pseudopeziza medicaginis*, highly resistant lines have been selected and are being incorporated into new varieties. Harvesting before much defoliation occurs and destroying plants in waste areas may reduce damage by removing sources of infection.

Yellow Leaf Blotch (*Pyrenopeziza medicaginis*)

This disease is present in all of the major alfalfa-producing areas of the world, but generally is less destructive than the pseudopeziza leafspot. In cool, moist weather, particularly if cutting has been delayed, the disease develops rapidly and damage may become quite severe.

Yellow elongated blotches develop parallel to the veins on the leaflets. Blotches often extend from the midrib to the leaf margin. Small orange to black fruiting bodies (pycnidia), barely visible to the naked eye, develop on the upper surface of disease areas, fig. 3. The function of the spores inside is not known. Diseased leaflets do not wither and die immediately, but after a period of wet weather considerable defoliation may result. Blotches which later become dark brown occasionally appear on the stems (*18*).

On dead leaves in late summer and fall, cup-shaped bodies (apothecia) form in abundance on the lower leaf surface. Small, oval, colorless spores are discharged from the fruiting bodies and are carried by wind to other plants.

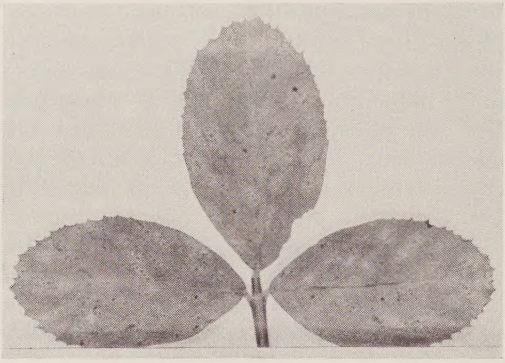


Fig. 3.—Yellow leaf blotch of alfalfa.

In late spring spores from overwintered apothecia supply the initial inoculum for infection.

Severity of the disease on the subsequent crop may be reduced by removing diseased leaves either by cutting or grazing before much defoliation occurs.

Stemphylium Leafspot

[*Stemphylium botryosum* (*Pleospora herbarum*)]

Leafspot caused by *Stemphylium botryosum* is a common foliar disease of alfalfa and less frequently of red clover. On alfalfa it is serious in wet years, particularly in dense stands. Spots on leaves are oval, and slightly sunken, dark brown with lighter centers, and usually surrounded by a pale yellow halo (Plate I, No. 3). Older lesions may be concentrically ringed. Usually a single large lesion causes the leaflet to yellow and fall prematurely. On red clover lesions are irregular, brown to black, and not ringed. Clover stems are seldom damaged, but the disease may attack petioles and cause leaflets to die (*37*).

The fungus overwinters on seed and in plant refuse.

Downy Mildew (*Peronospora trifoliorum*)

Downy mildew attacks alfalfa during wet or humid weather, particularly in spring. It occurs commonly on young plants, weakening them and causing defoliation.

Upper leaves are attacked first, developing a light green color; later they become yellow, twisted, and rolled. Large irregular-shaped necrotic areas also may develop on the leaves. A fine, gray, moldlike growth, consisting of mycelium and fruiting structures, develops on the underside of leaves. The fungus is systemic usually in new shoots, where it causes stunting and yellowing.

The numerous spores (conidia) produced on diseased leaves and shoots are disseminated by wind. In late fall resting spores (oospores)

are formed in diseased leaves, where they remain dormant during winter and germinate in spring. The fungus also survives the winter as internal mycelium inside the plant and on seed (*7*).

Where alfalfa is grown for dehydrating, Pennsylvania State University Extension workers recommend a fixed copper (50 per cent) spray as a control measure. Resistant lines of alfalfa have been found and are being tested for possible use in new varieties.

CLOVERS

Target Spot (*Stemphylium sarcinaeforme*)

Target leafspot is common on red clover in the northeastern region. The disease occurs throughout the growing season but damage is most evident in dense stands in late summer and fall, thus reducing the quality and quantity of the crop.

The fungus grows best at 68 to 75 degrees F. and requires high humidity for infection. It apparently does not attack alfalfa or other species of clover in the field. *Stemphylium sarcinaeforme* is seedborne and can survive on old plant parts or in the soil. Spores are splashed by rain or blown by wind to infect new leaves. The spores are hardy and can withstand drying for 18 to 20 months.

The leaf lesions are slightly sunken, round to irregular, and are characterized by light and dark brown concentric rings, fig. 4. If spots are numerous and coalesce, infected leaves turn dark brown, wither, and die (*15*).

Of many local and foreign varieties tested in a greenhouse at the U. S. Pasture Laboratory, none was highly resistant, but local adapted varieties, such as Pennscott and Kenland, were moderately resistant.

Powdery Mildew (*Erysiphe polygoni*)

Powdery mildew is one of the most common diseases of red clover, occuring on this host in the temperature zones throughout the world. Prior to 1921 the disease was uncommon in the central and eastern

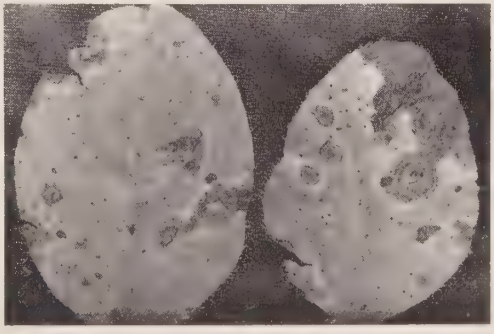


Fig. 4.—Target spot of red clover.

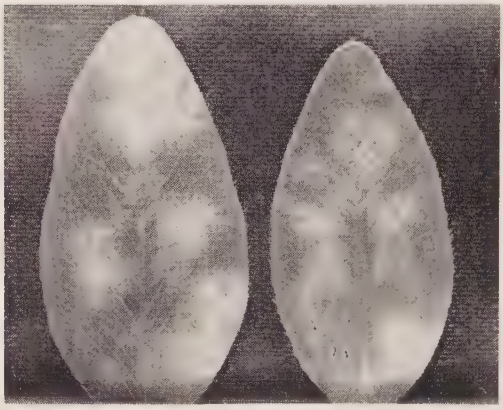


Fig. 5. — Powdery mildew of red clover.

United States, but within three years it became prevalent throughout North America. It can attack plants at any stage of maturity but develops best during late summer and fall. The disease is favored by relatively dry weather; frequent rains reduce its spread and development. Powdry mildew occurs on many hosts, including other species of clover. It is composed of many physiologic races, some of which may be restricted to specific hosts or groups of hosts. There is inconclusive evidence that the race or races of powdery mildew on red clover can infect alsike clover and several other species of *Trifolium* (*44*).

Symptoms consist of a light, gray to white, powdery growth of mycelium on the upper surface of a leaf, fig. 5. Infected leaves remain green for some time but ultimately lose their color, turning first yellow then brown and dry. Mycelium and spores are borne externally on the leaf surface, but specialized feeding strands (haustoria) penetrate the walls of host cells and develop inside. Numerous spores are produced on specialized stalks, the conidiophores, and are carried by wind and air currents to neighboring plants. The disease probably overwinters as dormant mycelium within the host.

Although European varieties of red clover have been reported to be more resistant than native American varieties, most American varieties show some resistant plants. Dusting with sulfur, while ordinarily impractical under field conditions, has been tried with some success in a few fields harvested for seed.

Spring Black Stem (*Phoma trifolii*)

This disease is termed "spring black stem" to differentiate it from "summer black stem," caused by *Cercospora zebrina*. It resembles the disease of the same name on alfalfa.

Spring black stem of clovers is caused by a group of fungi that are closely related but different somewhat, depending upon the host species attacked (*17*). The fungi involved include *Phoma trifolii*, the organism most frequently attacking red clover; and *Mycosphaerella lethalis*, cause of black stem of sweetclover.

The fungi responsible for black stem of clovers are widely distributed and may cause extensive damage during cool, wet weather in spring and fall. This is particularly true for sweetclover, where greatest injury usually occurs in the second year's growth. If the first 6 to 8 inches of spring growth become diseased, leaves and young shoots in thick stands may be so heavily infected that plants are killed.

Symptoms on petioles and stems consist of blackened areas which may ultimately spread over almost the entire plant. Small brown to black spots also occur on leaves. These usually enlarge and unite, causing leaf-yellowing and premature dying. Where seed is produced, the pods may be attacked and the seed become infected. Tiny, sunken, brown to black fruiting bodies develop in ashy-gray to light tan areas on mature stems or on dead stubble from earlier cuttings. The fruiting bodies contain masses of spores that infect new growth during periods of cool, wet weather. In northern parts of the United States the fungi overwinter on dead stems, the new shoots that develop becoming infected from the surrounding diseased stubble.

Since the fungi involved do not persist long in old crop residue, rotation is a practical control measure. Burning stubble before new growth develops in spring may also aid in reducing infection. Where early infection occurs, cutting or grazing earlier than normal may aid in reducing incidence of the disease on the subsequent crop.

Northern Anthracnose (*Kabatiella caulivora*)

Northern anthracnose is one of the major diseases of red clover in the cooler parts of North America, Europe, and Asia (*35*). In the United States the disease has been reported from Massachusetts to Minnesota and south to Delaware and Missouri. A similar disease, southern anthracnose, attacks red clover and other legumes farther south. Losses of 50 per cent or more have been reported in some fields. Hay production and quality as well as seed yield are seriously affected during periods of heavy attack. Northern anthracnose develops best during wet humid weather of spring and summer at temperatures of 68 to 77 degrees F. It does not attack alfalfa but has been reported on black medic, alsike, and white and crimson clovers.

Symptoms are confined chiefly to the petioles and stems, although leaflets are affected occasionally. Earliest symptoms on petioles consist of dark brown to black spots which become light-colored with dark margins. The lesions generally become $\frac{1}{4}$- to $\frac{1}{2}$-inch long and $\frac{1}{16}$- to $\frac{1}{8}$-inch wide and may girdle and kill the stem. The center of the lesion

frequently cracks. Lesions on petioles cause the leaf to wilt and the petiole to bend over in a typical "shepherd's crook" (Plate I, No. 4). Plants in fields heavily attacked look as though they were scorched with fire, inspiring the descriptive term "scorch," used to describe the disease in Britain.

Although seed has been infected artificially, there is no conclusive evidence that the fungus is transmitted on or in the seed. Presumably, the fungus overwinters in perennial green stems or in dormant tissues of the crown. There is also a possibility that the disease can survive an old infected stems. Wind and splashing rain carry spores from lesions to healthy plant parts. There is no evidence that the disease is soilborne.

Locally adapted varieties offer the best means of control. Seed from disease-free fields is also suggested as a precaution.

Southern Anthracnose (*Colletotrichum trifolii*)

This disease, similar to northern anthracnose, is caused by a related fungus, *Colletotrichum trifolii*. Some consider it to be the most destructive disease of clover in the southern states, where it can destroy stands completely (*32*). The disease occurs occasionally in the northeastern states and it has been reported as far north as southern Canada. Since the fungus is primarily a high-temperature organism (optimum 82 degrees F.), the areas of the Northeast most likely to be troubled by the disease are southeastern Pennsylvania, West Virginia, Maryland, and Delaware. The fungus also occurs on alfalfa, crimson clover, and white sweetclover; but it has not been observed on white clover. Alsike clover is considered to be almost immune. A conspicuous symptom on alfalfa is the presence of nearly white, dead shoots in late summer or fall.

While foliar symptoms are impossible to distinguish from those caused by the northern anthracnose fungus, *C. trifolii* frequently attacks the upper part of the tap root and crown. This results in a crown rot or tap root decay that weakens or kills affected plants. Diseased crowns often become so brittle that stems break off readily at the soil level. Tissues where the break occurs are usually discolored brown to bluish-black.

Spores of *C. trifolii* are generally straight while those of *Kabatiella caulivora* are usually curved. In addition the fruiting structures of *C. trifolii* contain black spines called setae which are visible with a magnifying glass.

Resistant strains of red clover have been developed in areas where the disease is prevalent. One of these, Kenland, is outstanding. Another, "Tennessee anthracnose resistant", is a locally adapted strain.

Fig. 6.—Pepper spot of Ladino clover.

Pepper Spot (*Pseudoplea trifolii*)

In early stages "pepper spot" is somewhat inconspicuous, but the damaging effects of the disease soon become apparent. Pepper spot is caused by a fungus which has apparently been active on clovers in the United States since before 1902 (*15*). Although the disease is most frequently observed on Ladino and white clovers, other species are attacked over a wide area in the humid, temperate parts of the United States. The fungus has not been reported on sweetclover as yet, but the same or a closely related species attacks alfalfa. "Pepper spot" is most damaging in dense stands of clovers.

During periods of cool, wet weather throughout the growing season, infected leaves and petioles are literally peppered with tiny, black, sunken spots caused by the fungus, fig. 6. The spots occur on both sides of the leaf and sometimes are so numerous that the infected leaves appear darkened or grayish. The tiny spots rarely enlarge, but their presence in great numbers seems to sap the strength of the leaf and it soon yellows and falls or withers and turns brown. The lower leaves in a thick stand of clover are most conspicuously affected and also are most severely damaged. Some plants are so heavily attacked during spring that they fail to survive summer drought or a severe winter. When flower stalks and floral parts are attacked, seed may become diseased.

Infection occurs from spores (ascospores) that develop in fruiting bodies (perithecia) on dead overwintered leaves and petioles. Infection spots occur on the first new leaves that emerge in the spring. As leaves die the organism again fruits and the spores infect new leaves that develop during the growing season.

No practical control measures are known, but observation has shown that plants differ in susceptibility, and selection for resistance is in progress.

Fig. 7.— Curvularia leafspot of Ladino clover.

Curvularia Leafspot (*Curvularia trifolii*)

A disease that sometimes causes considerable wilting and dying of leaves of Ladino clover is caused by the fungus *Curvularia trifolii*. The disease was first discovered in 1919 causing minor damage to white clover in the vicinity of Washington, D. C. Since 1940 it has occurred more frequently, presumably because of the widespread use of the more susceptible Ladino clover along the Atlantic Seaboard. Up to 20 to 25 per cent of the leaves may be attacked and damaged. Observation and tests suggest that Ladino clover is more susceptible than white clover. Although other species of clover can be infected under artificial conditions, they have not been found diseased in the field.

Infected leaves are usually distinguished by the presence of a large yellowed area that soon turns watery gray and translucent, then light brown. A yellowish band generally outlines the advancing edge of the lesion. In some cases, diseased areas that originate at a leaf tip become V-shaped, fig. 7. Sometimes the dead V-shaped part of a leaf curls downward. The fungus can invade the entire leaflet and grow down the petiole, causing wilting and killing of the leaves. The organism does not attack stolons (*27*).

The fungus is presumably spread by windborne spores. The disease develops most rapidly during warm, wet weather, a temperature of 75 to 80 degrees F. being most favorable.

Since the disease occurs mainly on leaves, grazing or clipping the clover when the disease is first observed will remove much of the infected tissue. This in turn reduces the amount of potential inoculum to infect new growth.

Cercospora Leaf and Stem Spot (*Cercospora zebrina*)

Cercospora leaf and stem spot, also called summer blackstem, is caused by the fungus *Cercospora zebrina*. The disease occurs extensiv-

ely on clovers throughout the northcentral and eastern United States and is sometimes severe on white clover in the South during summer and early fall (*15*). Similar species, *C. davisii* and *C. medicaginis*, attack sweetclover and alfalfa, respectively, causing leafspot and stem blackening. Damage varies from year to year depending upon weather conditions, but summer black stem is consistently present and frequently causes extensive shriveling of leaves and premature defoliation.

Symptoms vary somewhat on different legumes, but on clovers other than sweetclover the leafspots range from light brown to almost purplish-black. The spots on affected leaves are usually rectangular and lie between the veins of the leaf (Plate I, No. 5). When dew is still on the leaves, the lesions appear silvery from numerous spores produced by the fungus. Sunken lesions may nearly cover the stems of diseased plants. As spots on the leaves enlarge or merge, the affected areas shrivel and turn brown and the leaves either drop off or die prematurely.

On sweetclover and alfalfa large, circular, ashy-gray spots develop on the older leaves, which later shrivel and drop, fig. 8. On first-year stems of sweetclover, reddish-brown lesions sometimes develop in the fall after frost. The disease becomes noticeable on second-year sweetclover when plants begin to blossom. Occasionally the fungus is found early on shoots that are dying back following cutting or grazing. The fungus also attacks the flower heads, causing maturing seed to fall. Diseased seeds may be shriveled and discolored, or they may show no visible sign of infection.

The fungus persists on old stems, fruiting abundantly during warm wet weather. Infection is caused by spores disseminated by wind and rain.

Fig. 8.—Cercospora leafspot of alfalfa.

Removing old crop residues and clipping or grazing when the disease seems to be increasing may reduce damage to the next crops. Resistant varieties are being developed.

Sooty Blotch (*Cymadothea trifolii*)

Sooty blotch is found throughout the Northeast on most species of clover. It is most severe in low meadowlands.

The disease occurs during late summer and fall as olive-brown to black pustules which may almost cover the underside of the leaves (Plate I, No. 6). Spores are produced in great abundance on these mats of mycelium and are spread by wind and rain. Infected leaves at first remain green but tend to curl. Later they turn yellow, wither, and brown. In fall the overwintering stage of the fungus develops as raised, black, warty areas on leaves. These fruiting bodies (perithecia) contain spores which infect plants in the spring (*45*).

Leafspot (*Pseudopeziza trifolii*)

A leafspot of relatively minor importance except for occasional severe local outbreaks is caused by the fungus *Pseudopeziza trifolii*. The disease is similar to the common but more destructive leafspot of alfalfa caused by a different species of the same fungus. It is most important in the northern humid regions but is generally distributed throughout the United States, especially where moisture is not a limiting factor. The fungus attacks many species of clover, including white, red, alsike, crimson, and zigzag (*19*). A different species, *P. meliloti*, attacks sweetclover. Since it is the lower foliage that is usually attacked and destroyed, damage from the disease may be underestimated.

Small, brown, circular spots develop on either side of infected leaves. At an early stage the spots may be angular but they usually become round and vary in size from pin-point to $\frac{1}{8}$-inch in diameter. They generally remain separate and darken with age, becoming brown to almost black when mature. A distinctive characteristic readily seen with a magnifying glass is a small raised disk in the center of each spot. If the spot is moistened, the disk swells and is easily observed. The disks are fruiting bodies of the fungus and contain the spores that infect leaves of neighboring plants. Spores are shot upward and are disseminated by currents of air.

Rusts (*Uromyces spp.*)

Rusts generally attack clovers each year but damage is difficult to evaluate because infection usually occurs late in the season. Most of the newer improved varieties of clovers are attacked by rusts but probably not so severely as their predecessors, since very susceptible plants were either eliminated by nature or in selection programs.

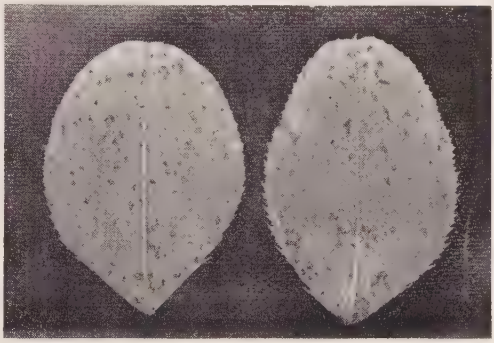


Fig. 9.—Rust of alsike clover.

Symptoms of rust on the different clovers are identical. A few pustules of the brown rust stage on the lower leaf surface usually cause no visible damage, fig. 9. If pustules are numerous and well developed, the upper surface of an infected leaf becomes reddish-brown to yellow. The leaf then curls or withers and dies. Petioles also may be heavily attacked and the supply of nutrients to the leaf curtailed. This hastens dying and reduces quality of the forage. Sometimes during fall or early spring, small, swollen, yellow clusters of tiny cup-like structures (aecia) develop on stems and petioles as well as on the midrib of the leaf. This is another stage in the life cycle of the fungus, and while it is usually less conspicuous than the brown rust form it causes distortion of infected leaves and petioles.

The rusts are differentiated by their host range (*1*). For example, the variety of rust on alsike clover (*Uromyces trifolii hybridi*) does not infect other clovers, while the rust of red clover (*U. trifolii fallens*) will attack zigzag clover, crimson clover, Egyptian clover, and several others. The rust on white clover (*U. trifolii trifolii-repentis*) does not attack red or zigzag clovers but does attack crimson and Egyptian clovers. Several of the most widespread clover rusts differ from the cereal rusts in the completion of the life cycle on a clover species without requiring an alternate host such as the barberry.

Infection occurs in late winter or early spring from thick-walled teliospores that overwinter on debris of diseased plants. Small secondary spores produced by the overwintered spores infect young leaves. From fruiting structures produced in the primary infection new spores are formed that spread the rust to neighboring plants, thus initiating the cycle for multiplying the disease. The spores are windborne and can infect plants under conditions of limited moisture, requiring only periodic heavy dews for germination of the spores. The spores can germinate over a wide range of temperatures.

Fig. 10.—Black patch of red clover.

Blackpatch (Unindentified fungus)

This disease was first recognized in 1933 in Kentucky, where it attacked red and white clovers. The disease was later studied in Wisconsin and more recently in West Virginia (*26*). It does not cause widespread damage but occasional outbreaks have resulted in losses in local areas. The blackpatch fungus attacks plants during periods of warm, humid weather in spring and midsummer. It can remain active and infect new growth during periods of drought, provided that heavy dews occur at night. The disease has been reported on red and white clover, soybean, cowpea, kudzu, and blue lupine.

The name "blackpatch" came from attacks by the fungus on scattered plants or groups of plants. All of the above-ground parts of the plant are affected, but symptoms on leaves and stems are most conspicuous. The lesions on leaves of red clover somewhat resemble those caused by target spot (*Stemphylium sarcinaeforme*). They vary from brown to grayish-black, usually in the form of concentric rings. Affected leaves generally wilt and droop, turning dark brown to almost black. Where fields are being saved for seed, greatest damage results from girdling of peduncles beneath the flower head or from direct infection of the flowers before seeds are fully developed. The fungus is seedborne and may cause seedling blight. Nothing is known at present concerning survival of the fungus on dead plant tissue or in the soil.

The fungus produces a coarse, dark, aerial mycelium which gives affected stems a fuzzy appearance, particularly when they are wet with dew or rain. At other times the fungus threads are readily seen with a magnifying glass, fig. 10. Since no fruiting structures have been found the fungus remains unidentified.

Seed treatment with a fungicide reduces initial infection. If severe foliar infection occurs, disease incidence on the next crops probably could be reduced by earlier harvesting. Crop rotation and deep plowing of diseased material also should be helpful.

Sclerotinia Crown and Stem Rot (*Sclerotinia trifoliorum*)

Crown and stem rot is caused by a fungus that attacks plants in winter and early spring at relatively low temperatures. It is one of the most serious diseases of alfalfa and clovers. Greatest damage occurs during mild winters or under snow cover. In the Northeast dead and dying plants are usually most noticeable from March to June, found either singly or in patches throughout a field. Most species of clover as well as alfalfa and birdsfoot trefoil are susceptible to the disease and in some years damage is severe. The fungus can also attack some 80 other species of plants, including certain weeds.

Plants of all ages are susceptible and symptoms vary accordingly. Earliest symptoms occur in the fall as small brown spots on leaves and stems. The leaves wither and die and the fungus spreads to the crown and upper root area. In late winter or early spring the crown or basal part of a stem becomes soft and discolored. Stolons of Ladino clover become soft and flaccid over small portions or over their entire length. As the plant parts die a white fluffy mass (the fungus mycelium) grows over the diseased area, forming hard, black sclerotia which may adhere to the surface of, or be imbedded in, the stem, crown, or roots (Plate I, No. 7). The sclerotia vary in shape and range from $\frac{1}{16}$- to $\frac{1}{4}$-inch or more in size. Sclerotia are the principal means by which the fungus survives and spreads. They can remain intact in the soil for several years (*21*).

From late September until snowfall when the weather is moist and cool, small ($\frac{1}{16}$- to $\frac{1}{4}$-inch diameter) flesh-colored, funnel-shaped fruiting bodies (apothecia) develop from sclerotia buried in the upper 1 or 2 inches of soil. Clouds of spores are shot from the apothecia and are carried by air currents to foilage of nearby plants, where they establish infection and repeat the disease cycle (*41*).

Longevity of the sclerotia in the soil makes the disease difficult to control. Planting seed free of sclerotia, clean cultivation, deep plowing to bury sclerotia beyond their capacity to develop fruiting bodies and long rotations aid in controlling the disease. Adapted resistant varieties being developed should aid in reducing damage due to this disease.

Root Rot (*Fusarium spp.*)

Root rot is one of the principal factors limiting production and maintenance of legumes. Depending upon environmental conditions, several different organisms may attack the root successively and thus become involved in the root-rot complex. Among the fungi most commonly associated with root rots of clovers are species of Fusarium, principally *Fusarium oxysporum*, *F. solani*, and *F. roseum*.

In the northern states stands of red clover are seriously depleted or even eliminated following the first cutting. External symptoms

develop first in the leaves, which curl at the edges, turn gray, and wilt. When the tap root of a diseased plant is cut lengthwise it is invariably discolored light brown to black. The discoloration sometimes occurs in localized areas corresponding to wounds on the root surface, or it may extend the length of the root in the central core (Plate I, No. 8. The tap root in dying plants is almost always completely rotted. New lateral roots are attacked as rapidly as they replace older damaged roots. This weakens plants and they succumb following mowing or during periods of hot, dry weather. The root rot fungi enter plants principally through wounds caused by root insects, winter injury, or mechanical injuries to the crown.

Control of root rot is difficult; however, adequate applications of lime and fertilizer enable plants to withstand infection. In addition, crop rotation and use of improved varieties are good farm practices that will help reduce the disease. Resistant varieties are being developed.

VIRUSES

The viruses that attack clovers also can infect many related and unrelated hosts. In turn, some viruses from hosts such as peas, beans, potatoes, alfalfa, and certain weeds are readily transmitted to clovers (*43*). Thus a virus-infected crop in one field or plants along a fence row may act as the reservoir for virus infection of an entirely different crop in a neighboring field. Spread and severity of infection frequently depend upon build-up in population of insects that carry viruses from plant to plant. Also important are the relative susceptibility of the host to the viruses present and the age of the plants when infected.

Most of our commonly grown clovers are susceptible to one or more viruses which are distributed widely throughout the United States. Symptoms of virus infection in the clovers vary somewhat, depending upon host species and the virus involved. In general, virus-infected plants have mild to severe mottling of leaves. There may be narrow, pale to yellow discolored areas along the veins or large light green to yellowish blotches between the veins of the leaf. In some cases leaves curl or are puckered or ruffled. Severely affected plants may be dwarfed or weakened and unable to withstand prolonged drought or severe winters. The weakening caused by virus infection may predispose plants to attack by other pathogens. Symptoms of most clover virus diseases are conspicuous during cooler periods, disappearing temporarily during hot weather.

Some examples of virus diseases of commonly grown clovers follow:

Red Clover.—Red clover vein mosiac is one of the most prevalent and widely distributed viruses of this host. The virus is identical

to Wisconsin pea stunt virus. In the field, red clover is also a natural host for yellow bean mosaic and potato yellow dwarf viruses. The viruses of alfalfa mosaic, pea common mosiac, and white clover mosaic can also infect red clover.

Alsike Clover.—Virus mosaic of alsike clover is widely distributed. In addition, alsike clover is known to harbor viruses of annual legumes, principally those of peas.

White clover (including Ladino clover).—In the field, white and Ladino clovers display at least two kinds of symptoms. Some plants develop vein clearing with mild mottling while others develop bright yellow patches or streaks between the veins of leaves. The vein-clearing and mottling symptoms are commonly caused by white clover mosaic, which consists of a mixture of two viruses, pea mottle and pea wilt. The virus causing yellow blotches between the veins, particularly noticeable in Ladino clover, is caused by a strain of alfalfa mosaic (Plate I, No. 9) (22). A similar strain of virus from alfalfa and Ladino clover causes a severe tuber necrosis of potato.

Sweetclover.—Many viruses can infect sweetclover in the field. For example, the following have been recovered from infected plants: alfalfa mosiac, yellow bean mosaic, several pea viruses, red clover vein mosaic, white clover mosaic, tobacco streak virus, and the Colorado rednode virus of bean.

To date, very little has been done to control virus diseases of clovers. Where a crop is being grown for seed, insecticides may be applied to reduce the number of insect vectors. Clover viruses rarely are seedborne. Where possible, fields of clovers should not be planted adjacent to other leguminous crops such as peas or beans. The ultimate solution is the development of varieties of clovers resistant to the most prevalent and damaging virus diseases.

BIRDSFOOT TREFOIL

Stemphylium Leafspot and Stem Canker (*Stemphylium loti*)

Stemphylium leafspot and stem canker recently has been recognized as an important disease of birdsfoot trefoil in the Northeast, particularly in New York State. Plants are partially defoliated and shoots are sometimes killed by girdling. Young leaf lesions are reddish-brown, slightly sunken, and round or semicircular. The spots turn darker and increase in size, becoming concentrically zoned. On stems the copper-colored cankers varying from dots to boat-shaped lesions 1 inch long, are outlined by a watersoaked border (Plate II, No. 1). The fungus also causes irregular brown discolorations on pods and seed.

Strains of the fungus on birdsfoot trefoil will not attack red clover or alfalfa and cultures from these plants will not infect birdsfoot trefoil.

The fungus can overwinter in raised black stromatic bodies on dead stems. Numerous spores are produced in the infected areas in the spring. The fungus is also seedborne (*10*).

LEGUMES AND GRASSES

Root Rot and Seedling Blight Complex (*Pythium sp., Rhizoctonia solani,* and *Fusarium sp.*)

Among the soilborne fungi that attack legumes and grasses, species of Pythium and Fusarium and *Rhizoctonia solani* are the most important. They occur throughout the world and attack a great variety of hosts.

Pythium debaryanum, P. graminicola, and *P. arrhenomones* are most commonly associated with injured grasses, while *P. debaryanum, P. irregulare,* and a few other species are pathogenic on the legumes (*13*). These species are most active in wet soil, some being favored by low, others by high temperatures. Several types of injury are caused by this important group of fungi. Seed decay occurs when seeds germinate slowly in cold, wet soil. Seedlings are sometimes damped-off before or shortly after they emerge. Seedlings thus affected wilt rapidly and fall over when a girdled rotted area develops on the stems at or slightly below the soil surface. Symptoms on older plants are less conspicuous and consist of blackened, rotted root tips, brown collapsed areas along laterial roots, or a general browning of the entire root system. Affected plants become pale green or yellow, are stunted and weakened, and usually die (*42*).

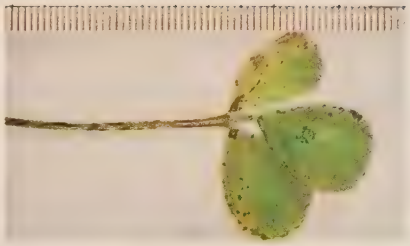
Although *Rhizoctonia solani* is present in most soils it is less destructive as a root pathogen in the Northeast than are the *Pythium* species. It causes damping-off and root rots of many legumes and crown or root rots of grasses. The fungus is most destructive when seedlings or older plants are weakened or are growing slowly because of unfavorable conditions.

Species of Fusarium damage legumes more severely than grasses. However, some species of Fusarium attack seed sprouts and roots of grasses, particularly under conditions unfavorable for the host.

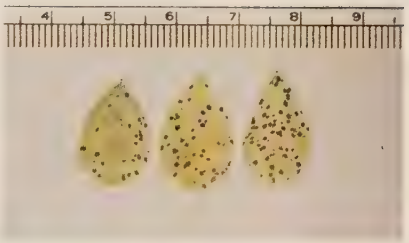
Fusarium damage to legumes is discussed in the section on clover disease.

Several practices can reduce losses from damping-off, seedling blight, or root rot. Adequate applications of lime and fertilizer and thorough preparation of seedbed enable seedlings to emerge rapidly and vigorously. Adequate applications of fertilizer also strengthen older plants and help them resist attacks by root-rotting organisms. In low, wet areas or in fields where seedling stands have failed, or where damping-off is known to occur, treating the seed with a fungicide may be helpful (*25*).

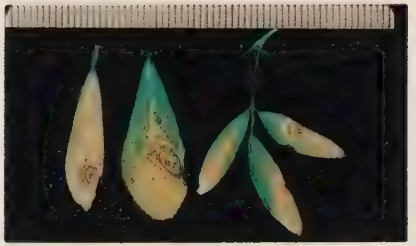
PLATE I.



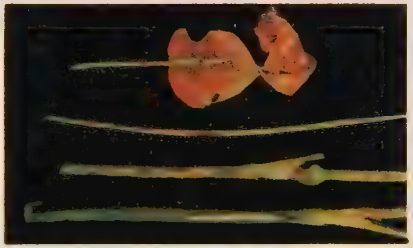
No. 1—Black stem of alfalfa



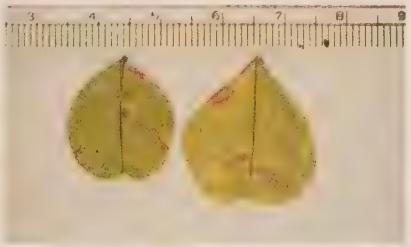
No. 2—Pseudopeziza leafspot of alfalfa



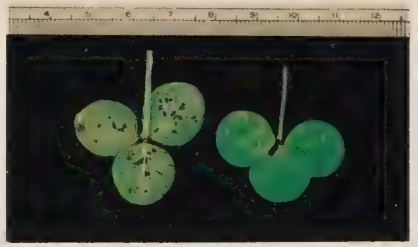
No. 3—Stemphylium leafspot of alfalfa



No. 4—Northern anthracnose of crimson clover



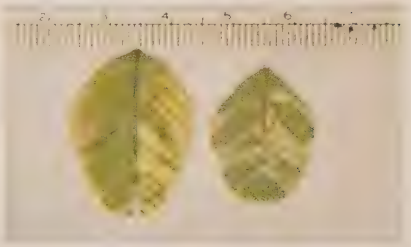
No. 5—Cercospora leafspot of Ladino clover



No. 6—Sooty blotch of Ladino clover

No. 7—Crown and stem rot—Sclerotia on alfalfa roots

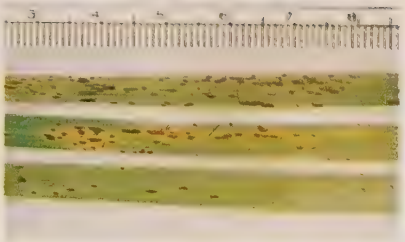
No. 8—Root rot of red clover



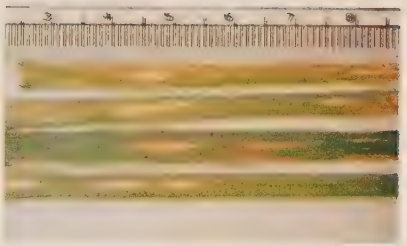
No. 9—Yellow patch virus of Ladino clover

PLATE II.

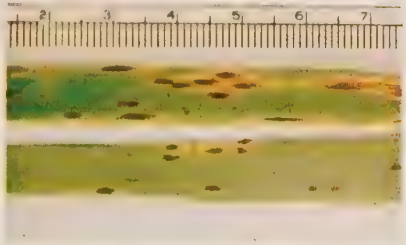
No. 1—Leafspot and stem canker of birdsfoot trefoil



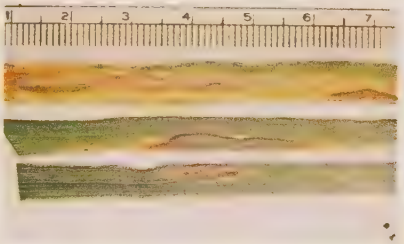
No. 2—Purple leafspot of orchardgrass



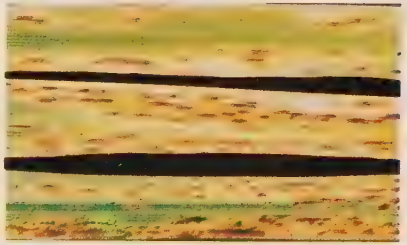
No. 3—Scald on orchardgrass



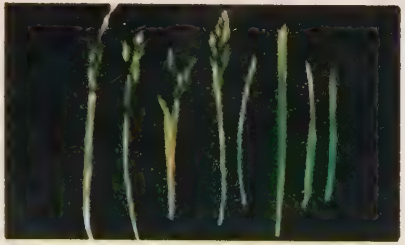
No. 4—Brown spot of bromegrass



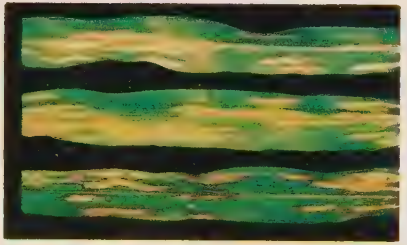
No. 5—Bacterial blight of bromegrass



No. 6—Net blotch of meadow fescue



No. 7—Bluegrass leafspot of Kentucky bluegrass



No. 8—Leaf blight of sudangrass

No. 9—Ergot on orchardgrass

Summer Blight (*Rhizoctonia solani*)

In addition to causing damping-off and various root rots, *Rhizoctonia solani* attacks the foliage of legumes and grasses (*7*). The disease is most destructive in dense stands during hot, humid weather. Plants are weakened and may be killed during periods of prolonged wet weather.

On birdsfoot trefoil, mycelium of the fungus grows rapidly over the foliage. Leaves become matted to the stems, shoots are killed, and large, sunken, straw-colored lesions with irregular brown margins are produced on the stems. These often girdle the stems and cause death.

Foliage of tall fescue, smooth bromegrass, and orchardgrass may be damaged by Rhizoctonia blight. Large straw-colored blotches outlined by irregular dark brown borders develop on leaf blades and sheaths. The lesions coalesce, causing death of leaves and sometimes of the entire plant. White to pale brown fungal threads can be seen with a magnifying glass on infected parts, and during rainly weather white tufts of mycelium may be visible to the naked eye.

Small tan to brown sclerotia sometimes develop on dead plant parts. These are structures which enable the fungus to survive for many years in the soil.

Since the fungus is soilborne and attacks a wide range of hosts, the only practical means for control is development of resistant varieties.

ORCHARDGRASS

Purple Leafspot (*Stagonospora maculata*)

Purple leafspot of orchardgrass occurs throughout the eastern United States. Lesions appear on leaves as soon as new growth begins in the spring, developing in the summer except during periods of prolonged hot, dry weather. The disease reaches a peak either shortly before or at time of heading and severely infected leaves are frequently killed. Even where orchardgrass is grazed, leaves become withered and brown from heavy attack by the fungus, and quality of the forage is reduced. Infection develops abundantly in the fall and new lesions can be found until snowfall.

The lesions usually appear as small, somewhat elongate, blackish-brown to deep-purple spots (Plate II, No. 2). When lesions are abundant they coalesce, causing the leaf to turn brown and die. Frequently the browning develops at the tip of a leaf or along the margin in long brown streaks. Small golden-brown bodies, the pycnidia of the fungus, develop within the dead areas of a leaf.

Infection occurs from spores that overwinter in pycnidia in dead stems and leaves. When spores emerge from the pycnidia they are disseminated by spattering rain or in wind-blown fragments of dead plant parts. The pycnidia are usually formed in rows in the dead parts

of leaves. In artificial inoculation tests in a greenhouse, infection occurred from 59 to 86 degrees F., with the optimum about 72 degrees (*11*).

Breeding for resistance apparently is the only practical means for controlling the disease. Although no immune plants have been found, sufficient resistance exists in some lines of orchardgrass to suggest they might be valuable for developing resistant varieties.

Scald (*Rhynchosporium orthosporum*)

This fungus, similar to *Rhynchosporium secalis*, which causes leaf scald of smooth bromegrass and cereals, produces distinctive lesions on leaves of orchardgrass. Although the disease usually is less prevalent and destructive than other leafspots, it causes damage when lesions are larger or when they are abundant and coalesce. In the Northeast the disease usually occurs during May or June when plants are growing rapidly. It sometimes occurs later in the season but seldom in autumn or fall. According to Caldwell (*4*), *R. orthosporum* is restricted to orchardgrass. Sprague (*39*), however, lists several other grass hosts as susceptible in the West.

Lesions on leaves are oval to elongate, light tan to white, surrounded by a tan to reddish-brown border (Plate II, No. 3). They vary in length from ¼-inch to 1 inch and may extend most of the width of a leaf blade. This may weaken the leaf, causing it to break over at that point. When the leafspot is abundant, the lesions occur in linear fashion on the leaf blade.

Infection probably results from conidia that overwinter on old dead leaves in the crown. In clonal nurseries of orchardgrass wide differences were observed with respect to resistance or susceptibility to the disease. It should be possible to develop resistant varieties if the disease becomes serious.

BROMEGRASS

Leaf Scald (*Rhynchosporium secalis*)

Smooth bromegrass, as well as other species of grasses and cereals, are sometimes severely damaged by leaf scald. The disease is most prevalent in the cooler humid and semi-humid regions of the world, having been reported in North and South America, Europe, Asia, and New Zealand. In the northeastern states the disease occurs on smooth bromegrass in late spring and autumn. In a moderately cool, wet season it may be found on leaves during much of the summer. The lesions occur most conspicuously on the leaf blade but may extend to the sheath. Caldwell (*4*) distinguished six specialized races of the fungus. The race on smooth bromegrass was restricted to species of Bromus. European studies, however, indicate that races from cereals can infect grasses and vice versa. Similar symptoms are produced by the fungus on all hosts attacked.

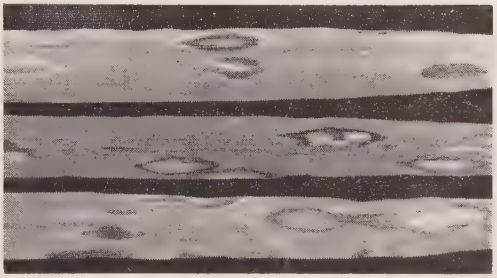


Fig. 11.—Leaf scald of brome.

Leaf scald first appears as dark bluish-gray, water-soaked blotches that become light gray with darker brown margins. Under favorable conditions the lesions enlarge and elongate until sizable areas of the leaf are affected and killed, fig. 11. This frequently results in premature death of leaves and reduces quality of the forage.

The fungus overwinters on dead leaves and old crop residues and in milder climates may produce new lesions throughout the winter. Spores are produced during cool, moist weather and are carried to healthy leaves by wind and rain. New spores are produced on old lesions as long as conditions remain favorable. The spores can germinate in water from 39 to 82 degrees F., but the optimum temperatures are 65 to 70 degrees.

Resistant lines of smooth bromegrass are available and are being used to develop varieties with superior characteristics. Elimination of old crop residues, crop rotation, and sometimes spring burning help to reduce infection.

Brown Spot [*Helminthosporium bromi* (*Pyrenophora bromi*)]

Smooth bromegrass is almost always severely damaged by brown spot. The fungus apparently attacks this host wherever it is grown in the northern United States and parts of Canada. The fungus attacks several other species of *Bromus* but no other grass hosts tested.

Infection is first noticeable as small, dark-brown, oblong spots on the first leaves which develop in the spring. Older spots are generally somewhat elongate, dark-purple to brown, and surrounded by a yellow band or halo (Plate II, No. 1). The lesions may coalesce, forming large yellowed areas on leaves. Severely infected leaves frequently die back progressively from the tip to the base. Brown spot develops best during cool, wet weather, reaching a peak in late May or early June. The disease spreads very little during the hot, dry weather of midsummer but becomes prevalent again in fall.

Primary infection occurs early in spring from spores discharged during cool wet weather (5). The spores are carried by air currents to new leaves where they germinate and penetrate directly through the

uninjured tissue. The fungus then becomes established in the host cells and produces characteristic lesions. As the lesions mature, spores of the fungus are produced either on the lesion itself or more frequently on the discolored parts of infected leaves.

Varieties resistant to brown leafspot are the best means for controlling the disease. Resistant lines are being investigated at several locations in the Northeast; these may ultimately comprise a variety that will reduce losses from this destructive foliar pathogen.

Bacterial Blight (*Pseudomonas coronafaciens var. atropurpurea*)

Bacterial blight, or chocolate spot, is widely distributed on smooth bromegrass, quackgrass, and several other grasses. The disease is most prevalent during periods of warm humid weather. Earliest symptoms on leaves consist of small watery spots with brown centers which later enlarge, become somewhat linear, and appear chocolate-colored or almost black (Plate II, No. 5). Lesions may coalesce and blight the entire leaf. Heads may become infected and the seed spotted. It is suspected that the bacteria may be seedborne. They can overwinter on dead leaves and stems (*33*).

Bacterial blight of smooth bromegrass is similar to halo blight of oats caused by a closely related bacterium. Although cultures of the blight organism from bromegrass can infect oats, the halo blight organism from oats does not attack smooth bromegrass. The bromegrass organism does not infect the other cereals.

Although this disease is not so serious as some on smooth bromegrass, it is capable of causing severe damage under some conditions. Resistant lines are known to exist and can be utilized if the disease becomes important.

TIMOTHY

Eyespot (*Heterosporium phlei*)

Eyespot is one of the most common foliar diseases of timothy and related species. In the United States the disease occurs primarily in the East and Midwest, and to a limited extent in the Far West. Eyespot has been reported on timothy in Europe and Japan and probably occurs wherever the crop is grown. Both Gregory (*12*) and Horsfall (*15*) observed heavy infection in fields of timothy in New York. Lesions may be so numerous that the leaves are killed prematurely, reducing appreciably the forage value of the crop. Although eyespot is most prevalent during the summer months it has been found on green leaves during almost any season. It has been suggested that the species of *Heterosporium* attacking timothy may be similar to or identical with species of *Heterosporium* on other hosts. In greenhouse tests cultures of the fungus isolated from timothy failed to attack orchardgrass, redtop, or Kentucky bluegrass.

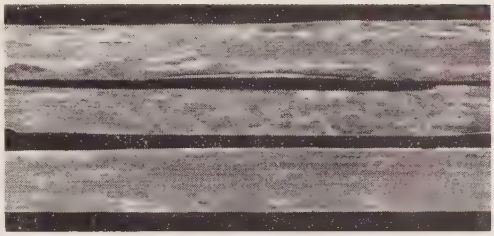


Fig. 12.—Eyespot of timothy.

Eyespot occurs on leaves as small, light-colored, oval lesions with a narrow violet border which later fades to brown, fig. 12. When lesions are abundant, the intervening tissue frequently becomes yellow. Affected leaves turn brown and wither prematurely.

Although infection probably occurs from conidia disseminated by wind and rain, it is difficult to find spores of the fungus on diseased leaves in the field.

Spores are capable of germinating at temperatures from 37 to 91 degrees F. (optimum 75 degrees) which may account for occurrence of the disease on growing leaves at almost any season.

No specific effort has been directed toward controlling the disease on timothy. Horsfall (*15*) found that fungicidal dusts were effective, but this measure would not be practical under most field conditions. The newer varieties of timothy are somewhat resistant to the disease.

FESCUES

Net Blotch (*Helminthosporium dictyoides*)

Net blotch is one of the most common foliar pathogens of tall and meadow fescues. It is so prevalent in the East and Southeast that the lesions are often used to distinguish the fescues from similar grasses. The net blotch symptoms on fescues resemble those on barley, caused by another species of Helminthosporium (*H. teres*). Leaf lesions, which are found throughout the growing season, range from small, oval, brown spots to extensive, brown, net-like streaks which may cause complete withering of the leaves and sometimes death of the plant (*7*) (Plate II, No. 6).

Spores are disseminated by splashing rain as well as by wind. The fungus is seedborne, which may account for occurrence of the disease in new seedings. In tests at North Carolina State College 3 to 5 per cent of the viable seeds from two Southeastern-grown samples were infected, while the net blotch fungus was not recovered from seed of one lot from Oregon.

None of several strains of fescue tested in a greenhouse was resistant. Differences in reaction among plants in both field and greenhouse, however, suggest the possibility of isolating strains resistant to the disease.

RYEGRASSES

Brown Blight (*Helminthosporium siccans*)

Brown blight is one of the more common diseases of Italian and perennial ryegrass in the eastern United States. On leaves of Italian ryegrass numerous small, elongated, dark brown lesions develop which may coalesce to form large, mottled, discolored areas. As many as a hundred tiny spots may develop on a single leaf and appear somewhat like net blotch on fescue, but the distinct net-like pattern has not been observed. Although these spots are less evident on perennial ryegrass, leaves of both species soon become yellow at the tip and gradually the whole blade and sheath withers and dies. Flower heads also may be attacked (*8*). During the past several years brown blight has not been of much economic importance and little work has been done on the disease.

BLUEGRASSES

Leafspot and Foot Rot (*Helminthosporium vagans*)

This fungus causes a leafspot and foot rot of Kentucky bluegrass which is probably the most prevalent and serious disease of this host in the Northeast. The disease occurs in lawns and pastures in the cooler, humid parts of Europe and North America and is most prevalent in the eastern and central parts of the United States (*8*). Pure, thick, stands of Kentucky bluegrass are more seriously affected than are mixed stands of grasses. Damage from the disease is accelerated by frequent close clipping since the succulent new growth is more susceptible. Application of nitrogenous fertilizers during summer stimulates formation of new growth, which in turn favors development of the disease. Damage is also severe when a heavy mat of clippings or hay is left on the ground during moist weather. Although *Helminthosporium vagans* is most destructive to *Poa pratensis*, it has also been reported on other species of *Poa* (*39*).

The leafspot appears as purple-black to reddish-brown lesions that vary up to ½-inch in length, depending on age of the lesion, susceptibility of the host, and width of the leaf. Older spots usually have a light center surrounded by a red to brown or purple margin (Plate II, No. 7). Some lesions extend the width of a leaf blade, causing it to break over or wither from the tip to the point of attack. The occasional development of paired, symmetrical lesions on opposite halves of a leaf blade indicates that a single infection occurred when the leaf was still tightly folded. Frequently the leaf boot is diseased, with the flower heads inside being attacked. Discoloration and blighting of all or a portion of the head results in infection of seed. If infection occurs at the base of a culm, it frequently spreads to the crown, causing browning of the tissues. This results in weakening

and death of the plant. Seedlings, particularly, are killed rapidly by stem infection. The disease develops best during cool, wet weather.

Infection occurs throughout the growing season from spores produced on older lesions. The spores are disseminated by wind and rain to neighboring healthy leaves. The fungus overwinters in lesions on living leaves or as spores or mycelium on dead leaves.

Except for applications on lawns or in park areas, chemical dusts or sprays to foliage are impractical as control measures. Further work is necessary to determine the extent of seed infection and whether seed treatment with fungicides is desirable. High clipping or mowing (1½ inches or more) reduces the incidence of foot rot by maintaining older leaves without inducing formation of succulent new shoots from the crown (*29*). Strains of Kentucky bluegrass differ in susceptibility to leafspot. Merion is the most resistant Kentucky bluegrass variety developed to date.

Septoria Leaf Blotch (*Septoria spp.*)

Although many species of Septoria attack grasses, two (*S. macropoda* var. *septulata* and *S. oudemansii*) are widespread and important on Kentucky bluegrass (*Poa prantensis*).

S. macropoda var. *septulata* was originally described from Spain on *poa pratensis* but either it or closely related forms have been collected in other parts of Europe, China, and Morocco. This disease, as well as *S. oudemansii*, is prevalent in the northern United States (*38*). Both diseases occur during periods of cool, wet weather, and consequently are most common in spring and fall.

On *P. pratensis*, *S. macropoda* var. *septulata* produces lesions at the leaf tips or along the leaf blade that are dark gray to brown and up to ¼ inch long. The lesions are sometimes bordered by red, maroon, or yellow bands. The fruiting bodies (pycnidia) may become prominent because color of the lesions often fades to straw or faint buff. *S. oudemansii* causes light purple to straw-colored lesions which may be small and scattered but are usually confined to the distal part of the leaf.

Infection occurs from spores that overwinter in pycnidia on dead leaves and old plant parts. The spores are capable of germinating over a wide range of temperature.

When the pycnidia become moist the spores ooze from an opening in the fruiting body and are held in a gelatinous mass. They are disseminated to neighboring healthy leaves by splashing rain and by wind-carried diseased plant parts.

Some reduction in disease can be achieved by removing or plowing under plant residues. Strains of grasses show differences in susceptibility and resistant varieties could be developed.

SUDANGRASS

Leaf Blight (*Helminthosporium turcicum*)

This disease is destructive on corn and Sudan and Johnsongrass, and is less serious on the sorghums. It is common on Sudangrass in the Northeast from midsummer until plant maturity, being particularly damaging during and after warm, moist weather. Many races of *Helminthosporium turcicum* from corn will attack Sudangrass, while in limited tests races of the fungus on Sudangrass did not attack corn. This may partially explain why Sudangrass is so diseased in the eastern states where leaf blight is prevalent on corn.

Large, elongated spots, tan to reddish-purple, often with dark margins, develop on the leaves (Plate II, No. 8). Later, lesions increase in size and fade to gray or straw-color, causing the leaves to appear blighted or scalded. Numerous pale brown, thick-walled spores are produced on the dead areas and are often visible as dark blotches. Flower heads of Sudangrass are also attacked and the seed becomes infected.

The fungus overwinters on plant debris. Results of field tests in several northeastern states showed that stands of Sudangrass were consistently increased by treating the seed with a fungicide. Control of *H. turcicum* may have accounted for a portion of this increase (*25*).

The varieties Piper and Tift are reported to be resistant to this pathogen as well as to the fungus causing anthracnose. Newer selections with even more resistance are being developed.

REDTOP

Red Leafspot (*Helminthosporium erythrospilum*)

Because redtop is a component of many grass seed mixtures, it occurs in many permanent pastures. Among the several pathogens that attack redtop is *Helminthosporium erythrospilum*, the cause of red leafspot. Although several other species of *Helminthosporium* have been found on redtop and related *Agrostis* species, *H. erythrospilum* seems to be the most common (*8*).

The disease appears wherever redtop is grown in the eastern and central United States, particularly following periods of warm, wet weather.

Lesions at first consist of small spots surrounded by a water-soaked area resembling a halo. The spots later become straw-colored with reddish or brownish borders of variable width. The lesions sometimes elongate rapidly, forming streaks. Infected leaves then wither and turn brown, particularly during dry weather. Sometimes withering without formation of definite lesions is the only sign of the disease, which then resembles drought injury.

Infection occurs from spores spread by wind and rain. The fungus overwinters on old dead leaves and infects new growth.

Little has been done to control the disease, but resistant plants are known to exist.

GRASSES—GENERAL

Brown Stripe (*Scolecotrichum graminis*)

Brown stripe, also called brown streak or brown leaf blight, is caused by the fungus *Scolecotrichum graminis*. According to Sprague (*39*), the fungus has been collected on more than 140 grasses and is widely distributed in the temperate zones of America and Europe. In the Northeast the disease is particularly severe on timothy, orchardgrass, tall oatgrass, redtop, and the bluegrasses. It also attacks the cereals and is commonly reported on rye. The disease is present throughout the growing season but is most evident during midsummer and autumn when leaves and culms are maturing. Severe attacks before maturity cause premature withering and dying of leaves. Quality of forage is appreciable affected and very susceptible plants probably are weakened.

On leaves, the fungus first produces circular to elongate, water-soaked spots which later turn deep, dull gray. As the lesions develop, their shape, color, and size depend somewhat on age of the plant and the host species attacked. Older lesions appear as narrow purple to brown streaks, usually with an ashy-gray to almost white center. The lesions may be ¼- inch to several inches long and occur singly or coalesce on the leaves and sheaths. Within the lighter center of each lesion prominent groups of black stromatic tissue and tufts of conidiophores develop. These appear as black dots in parallel rows along the surface, fig. 13.

Although several species have been described, most authors recognize *S. graminis* as the major form. Morphological variants have been isolated. According to Johnson and Hungerford (*16*), the fungus overwinters on dead leaves in tufts of grasses and infects new growth in the spring. No sexual stage has yet been conclusively demonstrated.

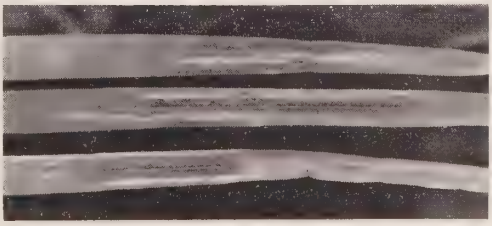


Fig. 13—Brown stripe of Canada bluegrass.

The broad host range of this disease, including such grass weeds as wild or foxtail barley (*Hordeum jubatum*), provides the fungus ample means for maintenance and spread. Burning dead grass aids in reducing overwintering inoculum, but development of resistant varieties is perhaps the best ultimate solution. Smooth bromegrass is one of the few grasses resistant to the disease.

Anthracnose (*Colletotrichum graminicola*)

Anthracnose is one of the most common and widely distributed diseases of forage grasses. It is particularly noticeable in late summer and fall on many grasses as they mature. Sudangrass, however, is attacked in midsummer at the height of vegetative growth. If conditions are favorable for development of the disease, the fungus attacks seedlings, causing stunting, wilting, and sometimes death. Although relatively few seedlings may be killed, damage to the root system in a diseased field usually results in material reduction in yield even though plants appear to recover as the season progresses (*3*). In older plants the culm or leaf sheath is attacked; however, the fungus may spread into the crown and roots of perennial grasses, frequently causing stands to die out in the second or third year. This is hastened in areas of low soil fertility. Early attacks cause a general reduction in vigor and premature ripening or dying with a resultant shriveling of seed.

Lesions that develop on the sheath or stem are usually light tan with a darker border of red or brown. Black acervuli of the fungus usually develop within the bleached center of a lesion or on leaf blades of dead plants, particularly when moisture is plentiful.

Infection occurs from diseased seed or from spores and mycelium that develop saprophytically on old crop residues. Infection of mature culms is found commonly in the vicinity of nodes. After infection is established and lesions develop, secondary spread occurs from spores or mycelium.

Colletotrichum graminicola is primarily a high temperature organism (optimum 82 degrees F.) which accounts for its association with maturity of grass in midsummer. Bruehl and Dickson (*3*) found a certain amount of host specificity among isolates. In general, isolates from warm season grasses, such as *Sorghum spp.*, were more pathogenic at higher temperatures.

General control measures include crop rotation, particularly avoiding a sequence of closely related crops; maintaining soil fertility; completely turning under plant residues; and development of resistant varieties.

Powdery Mildew (*Erysiphe graminis*)

Powdery mildew is most destructive on barley, wheat, and other cereals, but it also occurs on many wild and cultivated grasses. Some

of the more commonly attacked forage grasses are bluegrasses, fescues, orchardgrass, and bromegrass. The disease is generally not severe on grasses that are clipped regularly but it may reduce yields in seed fields. It is particularly troublesome on grass breeding stocks in the greenhouse. Disease development is favored by cool weather and, according to most workers, rather dry conditions.

Although many races of *Erysiphe graminis* have been reported, few are restricted to one species of grass. Also, since several races may attack a single plant, there is excellent opportunity for hybridization and formation of new races of the mildew fungus (*14*).

The fungus attacks the aerial parts of plants, but in most cases only the leaves are damaged. Oblong to irregular powdery blotches develop primarily on the upper leaf surface. Orange or yellow areas develop beneath the powdery blotches. The dusty, mildewy areas, first white, later gray or buff, may completely cover the leaf, causing it to yellow and turn brown.

The powdery masses consist of mycelium and numerous oblong, colorless spores borne in chains. Visible black fruiting bodies (perithecia) may develop in the spore clusters.

The fungus overwinters as mycelium on dead leaves and on winter-grown grasses. Perithecia are probably of secondary importance in winter survival. Disease symptoms are evident in late spring and the numerous spores are disseminated by wind to other plants. Perithecia containing ascospores are produced on dead tissue and probably serve to maintain the fungus over hot periods. With cooler weather in fall the disease may again become widespread (*6*).

While powdery mildew can be checked easily by dusting with sulfur, this is not practical except in small plots and in the greenhouse.

Ergot (*Claviceps purpurea*)

Ergot is a disease of world-wide distribution which affects the flowering parts of many cereals and grasses. The disease not only reduces seed yield and quality, but the sclerotia (resting bodies) contain a poisonous alkaloid which is harmful to animals. The horny, dark olive to purple sclerotial bodies (ergots) which project from the spikelets are the most conspicuous indications of the disease (Plate II, No. 9). These either fall to the ground when mature or are harvested with the seed. In spring with abundant moisture the sclerotia, whether sown with the grass seed or overwintered in the soil, send up tiny mushroom-like stalks with rounded heads which contain the fruiting bodies (perithecia) and spores. The thread-like spores ripen as the new grass crop blooms and are disseminated by wind and insects to the open flowers. Instead of normal formation of kernels, a sweet, clear, sticky liquid (honey-dew stage) containing millions of tiny spores exudes from the flower parts. Insects, rain, and wind disseminate this inoculum to

flowers on other plants. Later in the season infected ovaries enlarge and the fungus threads form the sclerotia (2). There are many races of the fungus, of which some are able to attack both cereals and grasses.

Resistant strains of various grasses have been selected, but sanitation practices are the best means of control at present. Ergot-free seed is the first requisite. Crop rotation and mowing of roadside grasses before the "honey-dew" stage is reached also will aid in the reduction of infection.

Snow Scald or Speckled Snow Mold (*Typhula itoana*)

This disease kills grasses and some cereals during winter and early spring under snow cover. It is most destructive on bent grass, ryegrass, and sometimes orchardgrass. As the snow melts, grasses attacked by the fungus become flattened mats of dead leaves and fungus mycelium. Diseased leaves are withered and turn brown to almost white. Numerous brown resting bodies (sclerotia), from $\frac{1}{16}$- to $\frac{1}{8}$-inch in diameter, develop on the dead leaves and stems.

The fungus survives summer months as sclerotia and in late autumn forms fruiting bodies bearing spores which infect other plants (*34*).

Fungicides applied in late fall have given satisfactory control of the disease on turf.

String of Pearls Disease (*Sclerotium rhizodes*)

Another foliar blight of grasses occurs during mid-to-late spring, particularly in low, wet areas of the northern United States and Europe. It has been reported on Kentucky bluegrass, timothy, redtop, and several other grasses.

Infected leaves often remain partially rolled or folded in the bud or are bent into crooks. They then become bleached and withered, and die. The most striking symptom is formation of white or brown sclerotia usually in a row along the diseases leaf; hence, the name "string of pearls disease." As the sclerotia mature they turn dark gray to almost black.

No spore-producing bodies are known for the fungus. Evidence indicates that the mycelium survives unfavorable periods in the soil and in plant parts (*40*).

RUSTS OF GRASSES

Stem Rust (*Puccinia graminis*)

Stem rust reduces hay and seed production of forage grasses as well as of the small grains. *Puccinia graminis* is divided into several sub-species which differ little in morphology but are specialized in parasitization of certain groups of grasses. In addition there are many

physiologic races within each variety of rust. In the Northeast *P. g. avenae* has been identified on orchardgrass, reed canarygrass, tall oatgrass and timothy; *P. g. phlei-pratensis* on orchardgrass, fescue, Italian ryegrass, and timothy; *P. g. poae* on bluegrass; *P. g. agrostis* on redtop.

The red rust or uredial stage develops on the leaves and culms of grasses any time during the growing season. As the rust infection develops, the leaf epidermis is broken and pushed back by the uredial pustules which contain golden-brown masses of spores. The spores are windborne and spread the rust to other plants. A distinguishing microscopic feature of the spiny urediospores is the presence of four conspicuous germ pores around the equator (*36*).

As diseased plants approach maturity, brown-black oblong to elongated telia (black rust stage) develop in the uredia or in new sori, generally on the sheaths and culms. The black teliospores overwinter and in the spring produce sporidia which infect barberry leaves. Two other spore forms, pycniospores and aeciospores, develop on the alternate host, the latter being capable of infecting the grass host. Windborne urediospores from other areas are probably an important source of infection.

Temperature and moisture determine the severity of stem rust. Low temperatures or alternate freezing and thawing are prerequisites for germination of teliospores. Moderate temperature (around 65 degrees F.) and moist conditions favor germination and infection by the urediospores.

In the Northeast, investigations concerning the economic importance of the grass rusts have been limited primarily to *P. g. phlei-pratensis* on timothy. In a study of parental clones at the U. S. Pasture Laboratory, severity of stem rust was correlated with the degree of winter injury. In Canada, where the disease markedly reduced the vigor and yield of timothy, strains were isolated which showed a high degree of resistance. The timothy varieties Milton and Marietta are reported to be resistant to stem rust.

It should be remembered that orchardgrass is a carrier of stem rust to oats. In addition, barberry should be eradicated within 200 feet of fields of orchardgrass and small grains.

Crown Rust (*Puccinia coronata*)

Crown rust is widely distributed in the United States, with specialized varieties and races on cereals and many wild and cultivated grasses. In the Northeast it is destructive to meadow fescue, but most strains of tall fescue are highly resistant. The variety of rust on fescue does not attack oats.

Crown rust causes considerable loss of leaves as well as reduction in quality of forage. Foliage of susceptible plants is usually killed.

On aerial plant parts bright yellow pustules develop and form powdery masses of spores. The black spore stage (telial) usually shows a ring around the pustules on the lower leaf surface. *Puccinia coronata* is most readily recognized by the crown-like knobs on the apical cell of a teliospore. The aecial stage, which is probably the main source of inoculum in the spring, is produced on an alternate host, the buckthorn (*Rhamnus spp.*). Other sources of infection are windblown spores from other areas and urediospores which survive relatively mild winters.

One collection of meadow fescue made in 1946 in Maine was immune to crown rust both in the field and in greenhouse inoculations (*23*). At present, clones of this collection being propagated at the U. S. Pasture Laboratory are no longer immune but are moderately resistant, indicating specialized races of the rust. Some backcross lines of these clones, however, appear to be immune.

Orange Leaf Rust (*Puccinia rubigo-vera*)

Orange leaf rust is made up of a group of specialized races and is found on many species of grasses (*28*). In the Northeast it occurs primarily on fescue, bluegrass, and redtop. The scattered orange-brown, powdery pustules form mainly on the leaves and occasionally on the floral bracts. Unlike *Puccinia graminis*, the leaf epidermis around the uredial pustules is not ruptured. Black telia arise independently of the uredia and remain covered by the host epidermis. A distinguishing morphological feature of the teliospores is absence of thickening of the apical cell wall.

Little is known concerning distribution and importance of *P. rubigo-vera* in the Northeast.

Yellow Leaf Rust (*Puccinia poae-nemoralis*)

Kentucky bluegrass is commonly attacked by yellow leaf rust wherever it is grown. This rust attacks several other grasses but does not infect cereals. It occurs primarily on leaves where it forms numerous orange-yellow pustules. Rust is most abundant on grass in undisturbed stands since frequent mowing or grazing removes many of the diseased leaves.

The rust has no known alternate host. Spores probably overwinter in uredial pustules on new leaves in the crown.

Leaf Rust (*Uromyces dactylidis*)

The principal leaf rust of orchardgrass is caused by *Uromyces dactylidis*. This rust occurs in mid-to-late summer and is most severe in southeastern Pennsylvania, Maryland, West Virginia, and states farther south. Macroscopically, the rust is indistinguishable from leaf rust caused by *P. rubigo-vera*, and both occur on the same alternate host (*Ranunculus spp.*) Microscopically, the two rusts can be dis-

tinguished by the one-celled teliospores of the *Uromyces* rust in comparison with two-celled teliospores produced by species of *Puccinia*.

Because of damage from lowered yield and quality of forage caused by this rust, efforts have been made to obtain resistant strains of orchardgrass. One such strain, selected at Beltsville, Maryland, should be available soon for use in areas where the rust is most serious.

The rusts frequently are parasitized by another fungus, *Darluca filum*. Many black fruiting bodies (pycnidia), visible to the eye, develop in the pustules and probably play a part in reducing multiplication of the rust. The spores of the parasite are colorless and two-celled with appendages at both ends.

SMUTS OF GRASSES

Stripe Smut (*Ustilago striiformis*)

Stripe smut occurs commonly on many grasses in the United States as well as in Europe and Australia. The important grasses attacked by one or more races of stripe smut in the Northeast are timothy, redtop, Kentucky bluegrass, and orchardgrass. Smutted plants can be found among grasses in many pastures from May to November. The diseased plants frequently die during hot, dry weather (22).

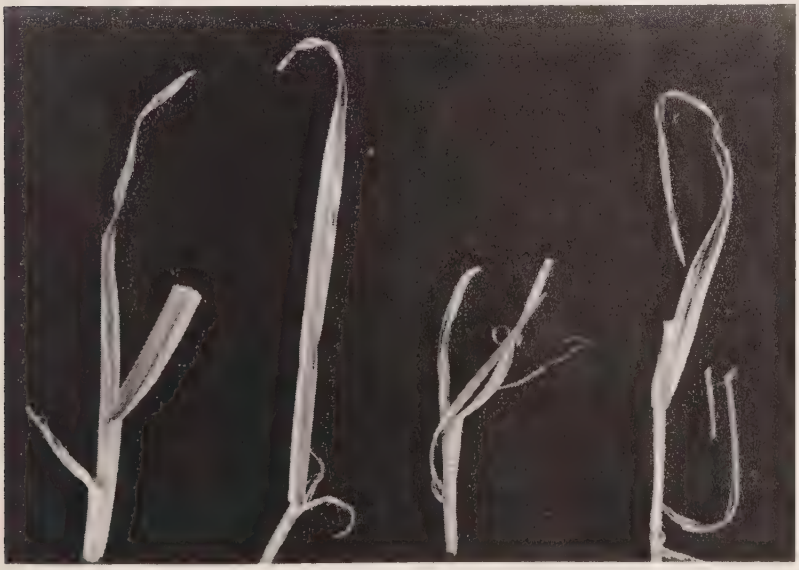


Fig. 14.—Stripe smut of Kentucky bluegrass.

Since smutted plants are usually stunted they may be easily overlooked in fields of uncut grasses. The disease is first noticeable as long, gray, narrow streaks which later coalesce to form dark stripes extending the entire length of the leaf blade. As the smut sori mature the leaf tissue is ruptured and the blades curl and become shredded, fig. 11. Smut spores released from the shredded leaves are carried to the soil by wind and rain (*30*).

Cool, moist weather in spring and fall are most favorable for development of the disease. Seedlings may become infected, presumably from seed contamination or from spores in the soil. Since the mycelium is systemic it can overwinter in the crown of perennial grasses.

Some attempts have been made to obtain resistance to the smut. However, because of difficulty in obtaining a high percentage of infection and because of a rather long incubation period for the disease, only limited progress has resulted. Treatment of contaminated seed with fungicides has also reduced primary infection. Other than rotation, there is at present no adequate means for eliminating the fungus from the soil.

Flag Smut (*Urocystis agropyri*)

Symptoms of flag smut, caused by *Urocystis agropyri*, are similar to those of stripe smut. Although this disease is usually not destructive it occurs on bluegrass, timothy, redtop, and quackgrass. A race of this fungus (formerly *U. tritici*) may be severe on wheat. Chlamydospores of flag smut can be distinguished microscopically from spores of stripe smut by the spore balls formed in the smut sori. The balls are composed of one to four dark, smooth, fertile spores surrounded by smaller colorless to brown sterile cells.

Black-Loose Smut (*Ustilago avenae*)

The fungus causing loose smut of tall oatgrass (formerly *Ustilago perennans*) has been identified as a race of *U. avenae* (*9*). Infection from the resting spores or from mycelium becomes established in the growing point. Smut sori develop and replace the floral parts.

Seed fungicides recommended for loose smut of oats would probably be effective if the disease became a limiting factor.

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GLOSSARY

Acervulus(i)—small, open fruiting body which becomes exposed by the rupture of the epidermis of the host, in which spores are formed vegetatively by abstriction from short, compact conidiophores.

Aeciospore—spores formed in chains within the aecium of a rust.

Aecium(a)—cup-shaped structure formed by the rusts in which the aeciospores develop.

Alternate host—one of two unrelated plant species required as hosts by certain rusts for the completion of their life cycles.

Apothecium(a)—open cup or saucer-shaped fruiting body whose concave surface is lined with asci.

Ascospore—one of several spores, normally 8, borne in a sac or ascus.

Ascus(i)—specialized cell or sac in which usually eight ascospores are formed as a result of the sexual process.

Canker—sunken area of dead or dying tissue in stems or roots.

Chlamydospore—thick-walled resting body formed by the rounding up of a segment of mycelium.

Chlorotic—deficient in chlorophyll, hence showing less than the normal amount of green color.

Conidium(a)—spore formed vegetatively by abstriction from a specialized branch of the mycelium, the conidiophore.

Conidiophore—specialized branch of the mycelium on which spores are borne vegetatively by abstriction.

Cross-infect—infection of one species of plant by inoculum produced on a different species.

Culm—the stem of grasses.

Fungus(i)—any member of that group of plants (excluding bacteria and Actinomycetes) which do not possess roots, stems, or leaves, and do not have the green pigment, chlorophyll.

Haustorium(a)—absorbing organ formed inside the wall of living cells, by means of which certain fungi derive nourishment from the cell contents.

Hypha(ae)—single strand or strands of mycelium.

Inoculum(a)—any entity, organism, part of an organism, or specialized structure of an organism which can induce infection in a host.

Lesion—area of tissue showing disease symptoms.

Life cycle—series of forms and relationships assumed by an organism from a primary stage to the next recurrence of that stage.

Morphology—study dealing with the form and structure of organisms.

Mycelium(a)—network of filaments or hyphae which make up an individual fungus plant.

Necrotic—dead or dying.

Oospore—immediate product of fertilization of an egg cell or oosphere.

Parasite—organism which derives its nourishment from and at the expense of another organism, the host.

Pathogen—disease-causing organism.

Peduncle—stalk which supports a flower or inflorescence.

Perithecium(a)—small, rounded, oval, or pear-shaped sexual fruiting body containing asci.

Petiole—leaf stalk.

Physiological race—pathogen indistinguishable from another in shape and structure, but which differs in its ability to attack certain varieties of the host; hence a pathogen which is morphologically similar, but physiologically different.

Primary infection—infection initiated by a pathogen following a period of seasonal inactivity.

Pycnidium(a)—small, rounded, oval, or pear-shaped fruiting body with a mouth or ostiole, and containing spores which are produced asexually.

Pycnium(a)—in the rusts, a small, flask-shaped structure arising from the mycelium which develops from a basidiospore, and in which pycniospores or spermatia are formed.

Pycniospore—minute spore or spermatium borne in the pycnium of the rusts, and functioning as a male cell.

Saprophyte—organism which derives its nourishment from dead organic matter.

Sclerotium(a)—resting structure formed by certain fungi, and consisting of a whitish inner zone of compact mycelium surrounded by a dark outer rind.

Secondary infection—infection resulting from inoculum produced by the pathogen during the primary cycle, or during other secondary cycles.

Seta(ae)—bristle-like projection.

Sorus(i)—spore containing bodies of the rusts and smuts which become exposed at maturity upon rupturing of the epidermis of the host.

Spore—specialized cell or group of cells adapted for dissemination and capable of germinating to form a new individual.

Sporidium(a)—one of several spores borne on a hypha (the promycelium) which develops from the teliospore of rusts and smuts.

Stromatic body—cushion-like mass of mycelium in or on which fruiting structures develop.

Systemic—spread throughout the tissues of the host; not localized or confined within definite boundaries.

Telium(a)—in certain of the rusts, the spore-bearing body or sorus in which teliospores are produced.

Teliospore—in the rusts and smuts, a spore which germinates to form a hypha called the promycelium on which the sporidia are abstricted.

Uredial—pertaining to the stage in the life cycle of a rust where urediospores are produced.

Urediospore—in the rusts, a binucleate spore capable of initiating secondary cycles by reinfecting the same host.

Virus—infectious entity capable of passing through bacteriological filters, and able to reproduce itself within the living cells of its host.

Yellows—condition of foliage resulting from destruction of the chlorophyll.

DISEASES ACCORDING TO HOSTS

Alfalfa

Birdsfoot Trefoil

Alsike Clover

Crimson Clover

Ladino Clover (White)

Red Clover

Sweetclover

Bluegrasses

DISEASES ACCORDING TO HOSTS
(Continued)

Bromegrass

Fescues

Orchardgrass

Redtop

Reed Canarygrass

Ryegrasses

Sudangrass

Tall Oat

Timothy